

Milk Fever

BY L. T. GILTNER¹

MILK FEVER, which causes the collapse and sudden death of high-producing dairy cows at the time of calving, is always accompanied by a serious unbalance in the mineral content of the blood. Treatment by a veterinarian, as described in this article, is an almost certain cure.

MILK FEVER, also called parturient paresis and parturient hypocalcemia, is an affection of cows occurring shortly after calving. It is characterized by paralysis of the motor and sensory nervous apparatus. Despite its name, milk fever usually is not accompanied by fever. There is always, however, a marked and rapid lowering of the blood calcium, so that the name "parturient hypocalcemia," which means too little calcium at calving time, is quite appropriate.

Milk fever is one of the most common, widespread, and serious of the acute afflictions of dairy cows. It affects almost exclusively the high-producing better nourished animals. It very rarely occurs at the first calving; sometimes at the second calving; and most frequently after the birth of the third to seventh calf, or during the prime of life. The poorly nourished cow is seldom a victim, and difficult calving (dystocia) is hardly ever followed by milk fever. After the first attack, the trouble may recur at one or more subsequent calvings.

CAUSE

The early investigations into the cause and nature of milk fever are of considerable interest to the present-day student for the light they throw on developments in medical science. Most veterinary writers today begin their discussions of the earlier studies of the disease with the work done late in the nineteenth and in the early part of the present century, when the theory of bacterial infection and systemic intoxication with bacterial toxins associated in some manner with parturition

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was in vogue. In 1897, Schmidt, a Danish veterinarian, acting on the theory that milk fever resulted from infection of the udder, experimented with injections of a solution of potassium iodide into the udders of cows sick with the disease. Although this treatment proved remarkably successful in his hands, it did not afford a satisfactory explanation of his infection theory, since he later found that when air was introduced into the udder along with the potassium iodide solution equally good results were obtained. In the early years of the present century it was found that oxygen or filtered air alone proved superior to the potassium iodide solution as a treatment. Bacteriologic investigations soon showed conclusively that milk fever was decidedly not a bacterial infection of the udder. However, Schmidt's treatment, or a modification of it—the injection of filtered air into the udder—proved so effective that no further investigations into the cause of the disease were undertaken at that time, since researches on other diseases were deemed more important.

An excellent treatment had been discovered, and when it was carried out skillfully, especially with strict aseptic precautions, milk fever could be readily controlled, or at least when cases occurred they could be cured. Research workers were apparently content to let matters stand, even though the underlying cause of the strange malady remained obscure.

Finally, however, in 1925, Dryerre and Greig² proposed the theory that milk fever is an expression of an acute deficiency of calcium in the blood. Studies of the blood of cows affected with the disease revealed that there was a marked fall from the normal calcium level. Moreover, when calcium in the form of calcium gluconate solution was injected intravenously in an appropriate dose into cows suffering from milk fever, a cure was promptly effected. Why, then, does inflation of the udder with potassium iodide, oxygen, or filtered air have a curative effect? Greig³ explains this as follows: "The effect is a mechanical one in that it elicits mammary distention and so prevents the further interchange of calcium from the blood to the gland acini [the microscopic saclike elements of the gland]. There is also reason to believe that the calcium, which is heavily concentrated in the gland, is forced back into the blood as the result of the mammary distention."

Chemical studies have revealed other abnormalities in the blood of cattle suffering from milk fever. In some cases hypermagnesemia (increase in blood magnesium) has been found, in others, hypomagnesemia (decrease in blood magnesium). A decrease in phosphorus and an increase in blood sugar (hyperglycemia) have also been observed.

In the normal animal all of the various blood constituents are maintained at a very constant level through the proper coordination of the body's complex and delicate glandular mechanism. The discovery of the changes in the blood brought about by the disordered functioning of this mechanism during milk fever has been of immense importance

² DRYERRE, HENRY, and GREIG, J. RUSSELL. MILK FEVER: ITS POSSIBLE ASSOCIATION WITH DERANGEMENTS IN THE INTERNAL SECRETIONS. *Vet. Rec.* 5: [225]–231. 1925.

³ GREIG, J. RUSSELL. THE NATURE OF MILK FEVER. 11th Internat. Vet. Cong. Rpt. 3: 306–324, illus. 1930.

in pointing the way to effective treatment, but the basic causes of the break-down of the mechanism have not yet been fully brought to light.

SYMPTOMS

The earliest stages in the development of an attack of milk fever are frequently not noted or pass unrecognized by the casual observer. The first symptoms, depression and disinclination to move about or to eat, may set in as early as 6 hours after calving, but more often they begin anytime from about the twelfth hour to the third or fourth day. Although milk fever symptoms may at times appear before parturition or even several weeks afterwards, such cases may be involved with other affections common to the parturient or post-parturient state, and the diagnosis should be confirmed by laboratory aid. Cases have been observed in which the animal appeared excited and highly nervous for a short time, with twitching of the muscles and jerking of the legs and head, but the classical picture of milk fever begins, as already stated, with depression, and this is followed by collapse and complete loss of consciousness. The animal lies with its head turned to one side, the eyes are dull and expressionless, the membrane covering the eye is reddened, the muzzle is dry, the extremities are cold, and the body temperature is usually below normal.

Udall ⁴ states that one of the most constant and characteristic signs in milk fever is a tonic spasm of the muscles of the neck, giving the neck a distinct lateral kink. This symptom is seen more frequently than the turning of the head to the side. Throughout an attack the animal ceases to feed or ruminate, and all body functions are at low ebb. The pulse and breathing are accelerated, the breathing often being labored and accompanied by groaning. Bloating is not uncommon. The most frequent complications of milk fever are pneumonia, which generally occurs as a result of inhaling food material regurgitated from the paunch while the animal is lying down, and septic inflammation of the womb from various causes. Animals that are not appropriately treated usually die within several hours to a few days.

PREVENTION AND TREATMENT

Among the serious cattle plagues, milk fever is unique in that its treatment when properly carried out is remarkably successful, while practical and effective preventive measures are lacking. Of the suggested means of prevention, the injection of a solution of a calcium salt directly after calving is worthy of mention, as is also the practice of partial instead of complete milking during the first several days after calving. Obviously, however, the former procedure is more or less experimental and not practical, and the latter may lead to mastitis. Special measures should be taken to insure an adequate ration for and proper care of the pregnant and parturient cow.

As already mentioned, however, there are two methods of treatment that are efficacious if properly carried out, but to insure success, treat-

⁴ UDALL, D. H. THE PRACTICE OF VETERINARY MEDICINE. Ed. 3, 672 pp., illus. Ithaca, N. Y. 1939.

ment must be instituted as early as possible. The procedure preferred by veterinarians consists in intravenously injecting calcium gluconate in 20-percent solution. It is sometimes necessary to repeat the injection one or more times to effect a cure. Solutions of other calcium salts may also be used, but care must be taken that the solution does not enter the tissues surrounding the vein, since this causes severe irritation, often leading to swellings or sloughing. The injection should not be made under the skin.

The alternate procedure, consisting of inflation of the udder with filtered air, must be undertaken with great care so as to avoid introducing infection by contaminating the teat canal. Equipment must be kept sterile and ready for use at all times. Before beginning the actual inflation, a clean sterile cloth is placed under the udder, the udder and the teats are cleansed with warm soapy water and dried with a sterile towel, and the teats and their orifices are disinfected. Each quarter is fully distended with air, and the teat is tied with a flat tape or bandage which is removed after not more than 3 or 4 hours. During an attack the animal should not be permitted to lie on its side, since there is danger of pneumonia from inhaling regurgitated paunch contents; bracing the animal so that it rests on its brisket will prevent this.

Usually with either the calcium-injection or the air-inflation treatment the response is rapid. In complicated cases the animal returns to consciousness in less than an hour and is often on its feet in 2 to 4 hours after treatment. The body temperature and the digestive and eliminatory functions return to normal, as evidenced by the resumption of rumination, defecation, and urination. In certain cases both procedures may be used, the one assisting the other in bringing about recovery. When medication in addition to the calcium salts is deemed necessary, the veterinarian always administers it by the vein, or hypodermically, and not by a drench, since there is danger that the medicine might pass down the windpipe and cause pneumonia. When treatments fail it is usually because there are complications, such as septic inflammation of the womb, pneumonia, or some serious involvement of the digestive organs.